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REPORT

Anaplasmosis

A Costly Disease Of Cattle

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ABOUT THIS REPORT . . .

Anaplasmosis has been long recognized as a major disease of cattle in this country. Few realize how great a toll it takes in cattle lives and beef and milk production.

Anaplasmosis is a disease of red blood cells resulting in anemia, emaciation, and frequently death. But no one is sure whether the organism causing it should be classified as a protozoan, virus, rickettsia, or bacterium. However, anaplasmosis most nearly resembles the protozoan diseases.

Loss estimates range as high as $50 million a year. Death losses are sometimes dramatic—for example, an estimated $4 million in Louisiana in 1958.

Since infected animals often appear normal, anaplasmosis may be present where it is unsuspected in a single herd or in many herds spread over a large geographic area.

In the past, lack of a practical test hampered diagnosis in anaplasmosis research. Then, as now, trade and management patterns of the cattle industry fostered perpetuation and spread of the disease.

Within the last few years, research has developed some important new techniques and information about anaplasmosis.

A practical complement-fixation serological test for diagnosis was developed cooperatively by the U.S. Department of Agriculture and several State agricultural experiment stations about 10 years ago. A number of laboratories around the country are now equipped to make this test.

A capillary-tube agglutination test has been recently developed by the University of Illinois.

Antibiotic treatments now being studied are encouraging.

Research has shown that individual cattle vary widely in their susceptibility to the disease and that calves are 10 times as resistant as mature cattle. Research has also shown that the disease can be spread by insects and ticks or by surgical and other instruments. Potential insect and tick vectors in different areas are being identified. The disease apparently is not spread by direct contact between animals. Some wild animals, including deer, are sometimes found to harbor the parasite.

Field studies using the complement-fixation test are gradually piecing together the geographic distribution and incidence of this disease. And some herd-management techniques directed against the disease look promising.

No effective vaccine for anaplasmosis has been developed.

Information in this report was provided by the Animal Disease and Parasite Research Division, the Entomology Research Division, and the Animal Disease Eradication Division of the Agricultural Research Service.

ANAPLASMOSIS, A COSTLY DISEASE OF CATTLE

Anaplasmosis is an infectious blood disease of cattle which takes a toll in lives of the animals and in production of beef and milk which is difficult to measure. The U.S. Livestock Sanitary Association has estimated the loss at $35 million in an average year. However, there is little research information by which to judge the cost of the disease other than in fatalities.

This has been known as a distinct disease for over half a century. It has doubtless been in this country for much longer than that.

The disease has been studied in various parts of the world for several decades, and much knowledge acquired, but important gaps remain—the classification of the organism causing anaplasmosis, the organism's life cycle, how to immunize against the disease, and how to save acutely ill cattle.

Great progress has been made, however, through research and field tests in the past decade. A practical specific test for the disease is an important tool available since 1954. Some specific herd-management techniques hold possibilities for cutting down on the disease and protecting animals from infection. And antibiotic treatments now under study appear promising.

DISTRIBUTION AND INCIDENCE

Anaplasmosis is a problem in about 25 States in the South and the West. It is not often recognized in other areas, although infection may occur occasionally.

In the South, anaplasmosis is concentrated along the coastal plain from Delaware to Texas, along the Mississippi River into Southern Illinois, and along the Louisiana-Texas border into Oklahoma.

In the West, anaplasmosis occurs widely in 11 States, but is concentrated in a Montana-Wyoming-Utah-Idaho area, an Oregon-Idaho-Nevada-California area, and along much of the California coast.

In various areas the incidence of the disease appears to fluctuate as the activity and population of insect and tick vectors increase and decrease with changing weather.

ARS and a number of States have been cooperatively blood testing cattle for anaplasmosis in most of the major problem areas. They have studied in parts of Mississippi, Tennessee, North Carolina, Kansas, Oklahoma, Wyoming, and Hawaii and to a limited extent several other States and gained a fairly good idea of the incidence of the disease there.

The accompanying map shows the distribution and the general incidence of the disease based on these surveys and on animal-disease reports from practicing veterinarians and others familiar with anaplasmosis. Significant numbers of infected cattle were found in all of the shaded areas, but not in every part of them. Some unshaded areas have scattered reactors.

For example, in Kansas, the eastern shaded area had 93 percent of the reactors found in the survey, but 7 percent were scattered elsewhere.

In Oregon, the disease is concentrated in the southeastern shaded area (Malheur County had 59 percent reactions to the test), but there are infected animals elsewhere, perhaps traceable to cattle shipped in.

In Wyoming, about 6,700 blood samples collected at random were tested for anaplasmosis. In 20 of the State's 23 counties, 13 percent of the cattle tested were reactors or suspects.

Anaplasmosis has been diagnosed in most of the counties of California, but the incidence has not yet been determined.

Hawaii's State veterinarian considers anaplasmosis to have been eliminated from that State through a cooperative State-Federal eradication program conducted there during the past 8 years.

EXPERIENCE IN INFECTED HERDS

The severity of anaplasmosis in a herd of cattle depends on a number of things—especially the virulence of the parasite, and the susceptibility of the cattle. Depending on the circumstances, the disease may be observed in the peracute, acute, mild, or carrier states.

Some farm experiences illustrate the various ways the disease manifests itself in herds.

In one case, a man purchased a ranch on the West Coast and brought in his herd from an uninfected part of the State. Ninety days later he lost three cows, found a fourth very ill, and called a practicing veterinarian. He found a large belligerant, 8-year-old Hereford cow, rigid, trembling, head high, and breathing hard, holding two herders at bay. She pawed the ground, ran a short distance, and lay down but after the cow was roped and treated at the owner's request, she struggled to her feet, lurched about 30 yards, collapsed, went into convulsion, and promptly died. This was an extreme case.

Other cattle became ill and were given citrated whole-blood transfusions without noticeable benefit. Within 2 weeks, six died during or after treatment, and three others were simply found dead. Nearby ranchers had been losing 25 to 50 head of cattle a year.

These are experiences with the *peracute form* of anaplasmosis in newly exposed adult cattle.

The *acute form* of anaplasmosis (the second example) is found from time to time in native cattle on many ranches of the same coastal area, as well as in any other enzootic area. An animal with a typical case is weak and usually nonbelligerent. It can be killed by overexertion, and must have gentle handling and good nursing care, such as plenty of water at hand. It can be driven to corral if rested occasionally on the way.

In an enzootic area, many of the cattle are exposed to the disease. Quite a few, though not all of them, will, sooner or later, have at least a *mild infection* (the third kind of case). A veterinarian, called on one such case, examined a weanling calf which was weak and depressed and had a slight fever. It seemed otherwise normal. A blood smear, when stained at the office, revealed anaplasma bodies in some of the red cells. Simple, gentle treatment was prescribed. During the next 90 days about

50 other calves showed similar symptoms and received the same care. All of the calves withstood the disease, as the young generally do, and in a few weeks appeared normal.

In enzootic areas such as those cited above, testing of a cross section of the cattle population generally reveals a substantial number of animals in the *carrier stage* (the fourth type of case). Although their blood is infective, few or no anaplasma bodies can ordinarily be found in a blood smear and no symptoms are evident. Carriers have the parasite, whether or not they have ever shown symptoms of the disease. Such animals are resistant to clinical illness as long as they retain the parasite.

TRANSMISSION

Natural vectors are the most important means of transmitting the disease. Many species of insects and ticks are capable of transmitting the disease in laboratory experiments. However, only a few of them—different species in different areas—are probably important natural vectors. Cattle may rarely transmit the disease to their young before birth, but not by contact with other cattle or through their feces or urine.

Anaplasmosis can be spread from infected to susceptible animals in two ways. Infected blood may be transferred mechanically by insects or on instruments used by man in dehorning, castrating, ear notching, tattooing, and other operations. Mechanical transmission takes place only when the disease agent is delivered promptly—that is within about 5 minutes. The perishability of the organism greatly limits the time and distance insects or instruments can carry the disease.

The other means of transferring the disease is biological. In this type of transmission, certain ticks feeding on infected animals pick up the organism with the blood they consume. The organism is able to survive for a long time in the tick's body and can infect other animals during subsequent feeding of the ticks.

Nineteen species of ticks have transmitted anaplasmosis under laboratory conditions.

In the West, ticks are thought to be the principal vectors, although the role of insects has not been adequately studied there. In the Intermountain region of the West the Rocky Mountain wood tick has been shown to be capable of transmitting anaplasmosis and it may be an important vector of the disease.

Along the California coast, the main vector appears to be the Pacific Coast tick. It lives in the mild climate of the low coastal ranges and feeds on cattle almost year round.

Ticks do not ordinarily move from animal to animal fast enough to transmit infective blood on their mouthparts. Instead, the disease agent remains alive for a long time in the ticks' bodies. ARS scientists recently found that the unmated male Rocky Mountain wood tick, which had fed on an infected animal, transmitted the disease to a susceptible animal after the tick has been exposed to a hibernating environment in a refrigerator for 6 months.

Some ticks can pick up the disease organism in one stage of their life cycle and transmit it in a later stage. In experiments, certain ticks have even passed it through their eggs to the second generation of ticks and from them to cattle.

Anaplasmosis is believed by many to be a particularly troublesome problem where ticks are important vectors. However, only a few infections developed when uninfected cattle grazed on Wyoming test pastures used 1 or 2 years previously by extensively infected herds. Moreover, there was little spread of anaplasmosis between infected and susceptible animals separated only by a fence in several experiments.

Wild animals that harbor the parasite in the blood may also be a factor in transmission in some areas. For instance, the Columbian blacktailed deer on the ranges and forests of the Pacific Coast is host both to the anaplasmosis-causing parasite and to the Pacific Coast tick. Such animals, which in themselves are hard to control, could be a persistent reservoir of the anaplasmosis-causing agent. For these reasons, we need to know more about wild-animal hosts in such areas and their relationship to the disease and to the vectors.

In the South, blood-sucking insects, particularly horse flies, appear to be chiefly responsible for spreading anaplasmosis. Nine species of horse flies have transmitted the disease in laboratory experiments. Stable flies, horn flies, mosquitoes are also abundant in the South but insufficiently studied as to their vector role.

Mosquitoes have transmitted the disease under certain experimental conditions, but their importance as vectors is not known. Further study of mosquito transmission is needed.

Of the 19 potential tick vectors of anaplasmosis, only 5 are common on cattle in the Southeast. Among these, the American dog tick may be the principal vector. The tropical horse tick, recently found in Florida, needs investigation. The blacklegged tick is an unknown factor. The Lone Star and Gulf Coast ticks did not transmit the disease in laboratory experiments.

In addition to the vector problem, an important contributing factor in the spread of anaplasmosis is the practice of shipping cattle for long distances. Long-distance hauling and concentration of animals at numerous points have provided ideal conditions for spreading the disease. Infected cattle carry the disease into noninfected areas. Noninfected cattle are exposed to the disease when moved into infected areas.

DESCRIPTION OF THE DISEASE

In the early stages of an acute infection, the body temperature may rise 2° to 5° F. and remain at 103° to 107° F. until the animal begins to improve. But if the infection is overwhelming, the temperature may drop below normal just before death. Heart beat may double in rate. Breathing may more than double in rate and become labored. As much as 60 to 75 percent of the red-blood cells may be destroyed.

When the red-cell numbers drop to as low as one-third of normal, the animals are in grave danger and usually die.

If the acutely infected animal survives the peak of cell destruction and the formation of new red cells exceeds the rate of destruction, convalescence usually follows and may continue for a month or two, but sometimes for a year or more. The period of convalescence is governed to some extent by the rate of red-cell generation, by the presence or absence of secondary complications, and by treatments given. Blood transfusions are sometimes given to aid the recovery of valuable animals. Young animals generally produce red cells faster than do older animals and also respond more satisfactorily to blood transfusions.

Relapses may occur after about 2 or 3 weeks of convalescence from an acute infection and the parasites build up for a while. Relapses may recur every few weeks for a year or more, as in malaria. This is just one of the characteristics of several protozoan diseases.

Aside from the occasional relapses during convalescence, the animal maintains a certain relative resistance to reinfection—that is, resistance to any new introductions of the parasite so long as some degree of infection remains. The resistance depends on the continued presence of the parasite rather than a lasting supply of protective antibodies such as many bacterial and viral pathogens stimulate.

Animals that survive infection become carriers. Although the animal shows no further signs of the disease, its blood is infectious. Such animals generally remain carriers for life and thus pose a threat to other animals. Any carriers freed of the parasite either naturally or through medication become fully susceptible again. Some exposed animals, especially the young, become carriers without experiencing acute anaplasmosis or having visual symptoms of the disease, since body defenses are able to hold parasitism to a minimum.

The disease agent, called a marginal body or anaplasma body, may appear near the margin of the red blood cells 15 to 45 days after the animal becomes infected. That body appears sphercial and is barely visible under standard microscopes. But as revealed by electron microscope it may have a long sac or tail. After becoming detectible in the blood, these bodies increase in size and double in number about every 24 hours for the next 7 to 11 days. The bodies remain at full size for 3 or 4 days; then they gradually disappear as the affected blood cells are removed from the blood stream by the animal's body. The severe loss of red cells in some cases is the main cause of death.

Most believe that the marginal body is the parasite and that its growth in size suggests a developing organism. In support, they cite electron-microscopic studies which indicate that the anaplasma divides into parts in the manner of a bacterial or protozoan cell.

On the other hand, no one has proved conclusively that the marginal body is the parasite, or what stage of the life cycle it represents. Marginal bodies are scarce and rarely seen in the blood of carrier animals. In experiments, blood serum from which all infected red cells have been separated was infective, even after having been passed through a filter fine enough to screen out objects larger than tiny bacteria. Moreover, no study, including biochemical analyses of parasitized blood, has conclusively shown whether the parasite is a bacterium, a virus a protozoan, or a rickettsia.

In addition to the type of anaplasmosis that infects cattle but no other domestic animals, there is another type of anaplasmosis which infects sheep and goats, but not cattle. This disease is mild and apparently rare in this country.

DIAGNOSIS, TREATMENT, AND HERD MANAGEMENT

Diagnosing the disease became less of a problem when a laboratory technique became available about 10 years ago. More recently, methods of treatment and herd-management have added to knowledge about coping with the problem.

Diagnosis by observation of the animal has been handicapped by the similarity of certain other disease symptoms, but there are some useful signs. Excessive urination and constipation, especially when occurring together, suggest advanced anaplasmosis. Jaundice is common in such cases. Acute anemia in young animals may result from several diseases, but in an anaplasmosis area, acute anemia in older cattle suggests anaplasmosis.

The complement-fixation test is an invaluable aid in diagnosis. It gives a high degree of accuracy in diagnosis and often can detect new infections before the animal becomes visibly ill.

A new serological test (the previously mentioned capillary-tube agglutination test) for anaplasmosis is being studied. However, the practicality and reliability of the test have not yet been fully determined.

Treatment

Considerable study is now under way to determine the effectiveness of antibiotics in the treatment of anaplasmosis. These studies are being pursued along preventive as well as therapeutic lines. Although preliminary results from the use of antibiotics are encouraging, these investigations have not developed to the point where conclusive evaluation can be made. In considering treatments, the local veterinary practitioner should be consulted and the measures carried out under his supervision. This is particularly important when dairy cattle are involved, for drug residues are not permitted in milk.

Several supportive treatments for sick animals have come into use. Transfusions of 2 to 3 gallons of citrated whole blood have been used with variable results. Blood transfusions may hasten recovery in early acute or subacute cases if the animals can be handled without excessive excitement. It is doubtful that transfusions save many gravely sick animals from death. Certain arsenicals have some value in stimulating the production of red blood cells and correcting anemia. Glucose injections help restore body moisture and energy. Laxatives are sometimes used to relieve constipation. Simple, good nursing is often prescribed without treatment for weak and anemic animals which cannot tolerate the exertion of restraint for treatment.

Management Measures

Each year, thousands of cattle owners face the problem of what to do about anaplasmosis. Fortunately, research has opened a few courses of action not understood just a few years ago. But the condition of the animal, the herd, and the area environment determines what, if any, recourse is practical. The effects of management practices on anaplasmosis incidence and spread have been measured in a number of States through Federal-State cooperative field studies.

First, consider the fairly typical experiences of two cattlemen who discovered anaplasmosis in their herds but did not change management practices or treat the animals.

Some indications of anaplasmosis infection were first noticed in a Louisiana herd a few years ago. By the spring of 1958, when vectors were abundant, the cases became more evident. At that time the 240 animals of the herd were tested and 32 percent reacted positively. Visible symptoms of the disease showed up in the herd during the summer, and 67 percent reacted when tested the following winter.

A Missouri herd of 550 cattle with a similar background had 18 percent reactors when first tested in 1955. Visible symptoms of the disease continued to show up in the animals and 46 percent reacted when tested 3 years later.

Testing and sale of reactors for slaughter, on the other hand, have been effective in some areas in cleaning up herds with a substantial rate of infection. A Tennessee cattleman, who lost 60 animals due to anaplasmosis in 1957, had the 266 survivors tested. One hundred and fourteen tested positive or suspicious and were sold for slaughter. This plan was continued, with minor losses, the next year. And on the fourth retest 2 years later, only 2 suspects and no reactors were found.

This method was also effective in cleaning up a moderately infected herd on a restricted range. A Montana experimental herd of about 325 animals was isolated each winter but grazed each summer on a range separated from infected herds on three sides by only a barbed wire fence. Although the experimental cattle showed no visible symptoms in 1953, a test showed that 5.5 percent of them (18 head) were positive reactors or suspects. Before removal to summer pasture each year, the herd was retested, and, except for one bull, the reactors or suspects were slaughtered. That bull deviated between weak positive, negative, and suspect status through the first year and was then sold. The successive yearly herd tests showed 1, 4, and 2 suspects only. The abundance of natural vectors and the proximity of infected cattle did not interfere in cleaning up the herd.

To further study this method in an enzootic area, 15,000 cattle in 79 herds in the Mississippi-Yazoo River Delta were tested and separated into two groups on each farm—one herd of reactors and the other herd of nonreactors. In the preceding year, 659 cattle in those 79 herds had died of anaplasmosis, and 2,248 showed symptoms of the disease. The insect-vector population was high.

At the start of the study, the cattle were tested twice in the spring and found to have 50.5 percent reactors. However, the disease rate varied widely from herd to herd despite the uniform geographic character of the area.

Test-negative cattle in most herds were then pastured separately from the reactors, but a few owners disposed of all negatives, and a few owners disposed of all reactors. Despite the late segregation of the cattle after the spring insect buildup, the disease rate in the segregated negative herds was only 10.8 percent by fall, and the number of fatalities was restricted to 14 cattle. However, a 50-percent decrease in insect population that year contributed to the low rate of spread.

OUTLOOK FOR GETTING RID OF ANAPLASMOSIS

The prospect of controlling anaplasmosis effectively depends on knowledge and development of technology, on vectors and other influences in the environment, and on production and marketing practices of the cattle industry.

In the realm of technology, the complement-fixation test tells us where the anaplasmosis problem is, and which herds and which animals are infected. The complement-fixation test is now being used in diagnosing acute and carrier cases in certain farm herds and used in field studies and research. Testing is indispensable in efforts to reduce the toll from the disease.

The role of antibiotics in prevention and treatment of anaplasmosis is being studied. Depending on future developments, antibiotics may have a definite place in controlling the disease.

Many scientists and others consider the development of an effective vaccine for this disease one of the top research needs. Attempts to develop a vaccine have failed, partly because it has been impossible, to date, to grow the parasite outside the body of its natural host animal. The prospects of developing successful immunization techniques are not encouraging. The fact that fully recovered animals which no longer harbor the organism are fully susceptible is discouraging. In other words, even an attack of the disease does not confer immunity, but only a resistance as long as the carrier state persists.

Other important technological needs—classification of the parasite and determination of its full life cycle—are unfilled to date. These may depend on developing the means of keeping the parasite alive and reproducing outside the host animal, so it can be studied in detail. The work done to date has provided few leads into the problem.

The good results from isolating infected animals from healthy ones in field trials and our recent knowledge of vectors suggest that such isolation plus vector control may be a practical means of ridding herds of anaplasmosis in some areas. Success of this method would depend on avoiding conflicting problems in such matters as introduction of infected herds into the area for seasonal grazing. Success would also depend on buying only those replacement animals testing negative or those from noninfected areas. But at best control under range conditions is difficult.

Overcoming anaplasmosis involves the individual animal, the herd, the community, the State, the Region, and the Nation. Three plans have been followed in field trials in selected areas of the South where livestock owners asked for the help of USDA and the States. To insure reliable evaluations, to conserve technical facilities, manpower, money, and materials, and to prevent further spread of the disease, the participating farmers agreed to carry out one of several specified procedures after the first herd test:

Plan I. *Owners whose herds have a low incidence of the disease* usually select this plan and promptly sell all reactors for slaughter. This is the plan used in Hawaii's successful eradication program which began in 1955.

Plan II. *Owners whose herds have a relatively high incidence of the disease* usually choose Plan II and segregate their reactors from the negative animals, with the understanding that the reactors will be sold for slaughter as soon as practicable.

Plan III. *Owners whose herds have a high incidence of the disease* may elect to test the heifers at weaning time and retain all the negatives separate from sources of infection to build a clean herd. Most calves from infected mothers are born free of infection, and about 85 percent of them test negative at weaning. This plan may result in an anaplasmosis-free herd within a few years.

Although it is not expected that these control plans will work well everywhere, it is conceivable that communities may wish to organize to follow one of these plans cooperatively where considered feasible by experienced veterinarians. Tick vectors and possibly wild-animal reservoirs of the parasite may present difficult obstacles in some areas. Range management of herds involving limited or few contacts with the stock also constitutes a problem. It will require much research to adapt control plans to usual range management situations.

ANAPLASMOSIS

General Distribution and Incidence

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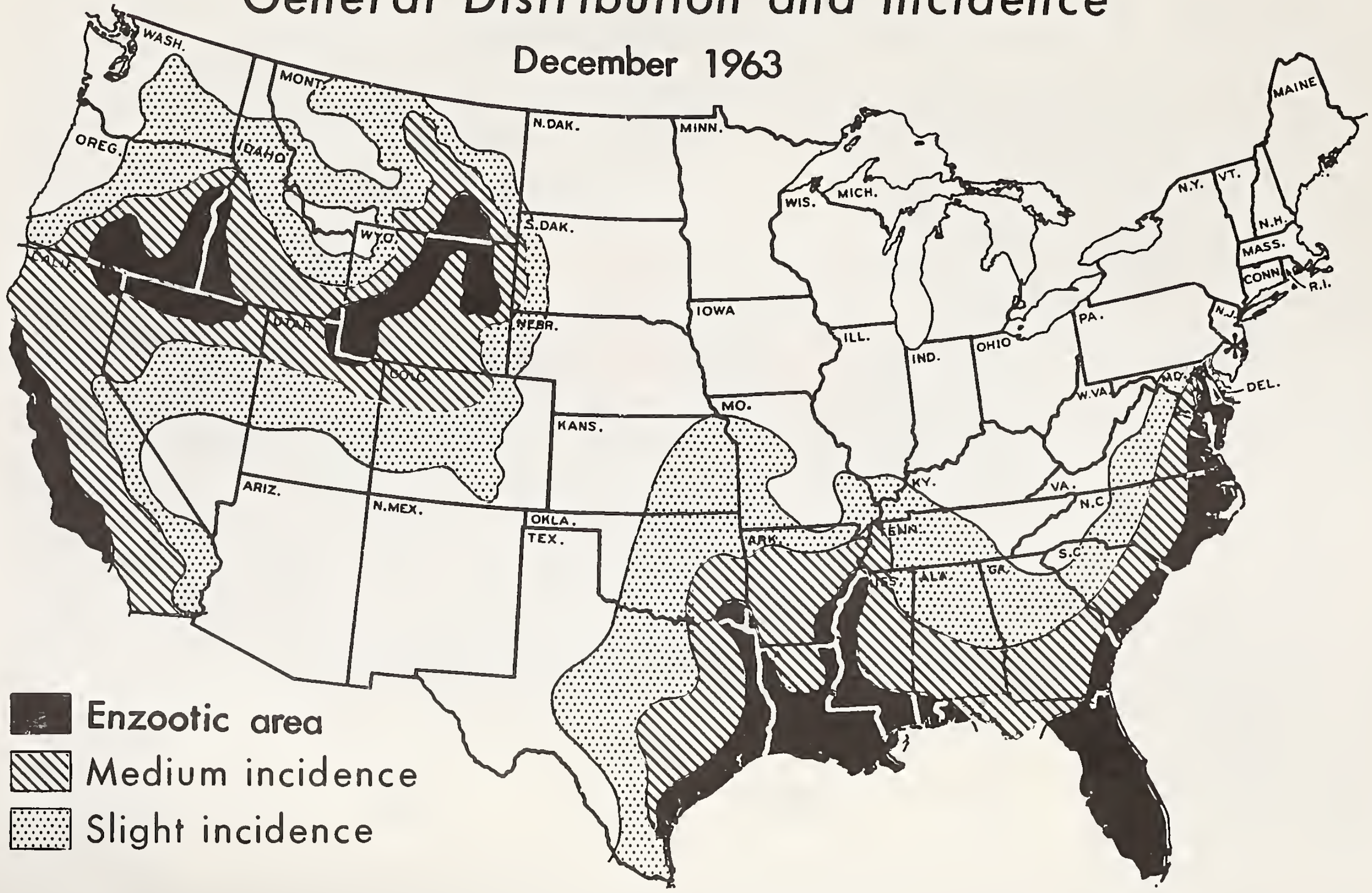

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